ACUTE SEPTIC INFECTION OF THE THROAT AND NECK; LUDWIG'S ANGINA.

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THE acute septic infection which involves the mouth, throat, neck, submandibular and parotid regions, known clinically as Ludwig's angina, is as yet not thoroughly understood as regards its pathology, nor is its treatment efficient. It is an extremely fatal disease, and in many instances probably unnecessarily so. The modern exact methods of observation, record and research should be applied to it, so that the affection can be recognized and its treatment placed on a proper scientific basis. Septic inflammations of the neck were more or less well known before 1836, but in February of that year D. Ludwig, of Stuttgart,[1] described what has since been known as Ludwig's angina. He stated that it was fatal in almost all cases. It began with slight fever, chills, headache, disturbed appetite, coated tongue, and often difficulty in swallowing. Usually on one side of the neck affecting the cellular tissue in the region of the submaxillary gland, rarely the sublingual or parotid, a hard swelling appeared. It spreads under the chin around the neck to the opposite side, over the larynx and perhaps the parotid. The sublingual region is infiltrated and the tongue rests on a hard base. It is both painful and difficult to open the mouth. Speech is impaired and partly from pressure on the larynx and partly on account of invo vement of the smaller neck muscles the voice is rough and guttural. Mucus, which is difficult to expectorate, accumulates in the throat.

Early in the disease, during the first four or six days, the skin is not red and the constitution not much affected. Later openings occur posteriorly on the inside of the mouth, and a thin gray or red brown evil-smelling liquid exudes.

A gangrenous odor develops, the lungs become affected and death ensues in ten or twelve days. On post-mortem examination the cellular tissue and muscles around and under the jaw and the posterior portion of the throat are found to be gangrenous.

This description of Ludwig is typical of the severer forms of the affection, and it was henceforward known as Ludwig's angina.

In 1895, however, Felix Semon[2] of St. Thomas' Hospital, London, in a paper before the Medico-Chirurgical Society, claimed that the various affections hitherto described as acute œdema of the larynx, œdematous laryngitis, erysipelas of the pharynx and larynx, phlegmon of the pharynx and larynx and angina ludovici were simply various forms of acute septic inflammation of the throat and neck and pathologically identical; also that they merely represented degrees varying in virulence of one and the same process, and that the question of their primary location and subsequent development depends in all probability upon accidental breaches of the protecting surface through which the pathogenic micro-organism finds entrance; and that it is absolutely impossible to draw at any point a definite line of demarcation between the purely local and the more complicated, or between the œdematous and the suppurative forms. His views have been more or less accepted by probably the greater number of writers on the subject.

Before a disease can be said to be mastered we must understand its pathology and therefrom deduce a rational method of treatment. Any method of treatment not based on the pathology of an affection must be more or less empirical and therefore to a considerable extent unreliable and uncertain. For this reason a knowledge of the pathology of a disease is the first step toward efficient treatment. Ludwig recognized that the disease was one of septic infection, but in his day, 1836, bacteriology was practically unknown, and he was limited in his knowledge to clinical observation and gross post-mortem examination. We

should know (1) what is the germ or germs that start the infection; (2) how do they gain access to the tissues; (3) what tissues are attacked; (4) how the infection progresses; (5) how it influences the parts locally and, finally, (6) how it affects the system generally. Our knowledge is so incomplete that partial answers only can be given to these questions.

1. What is the germ or germs that start the infection?

Inflammations of the throat can be produced by mechanical and chemical irritants, as by injuries and poisons and œdema occurs in Bright's disease, but these are not due to infection and as a rule do not resemble the latter either in their clinical appearance or course. As regards the character of the infection we are still considerably in the dark. In almost all cases germs are readily detected, but their exact nature and action are to a great extent unknown. In some cases pure cultures of a single micro-organism are found while in others so many are present that it has been found to be impossible to identify them. Even when only one or two kinds are found it is not proof that others were not present likewise. Mixed infections are common. If crepitation is present in the tissues it is assumed that a gas-producing bacillus is its cause, and while it may be found it will probably be accompanied by other organisms. Fetor is likewise attributed to a bacillus, but this is not so certain as in the case of gas. Fetor is a common accompaniment of severe cases, yet the presence of bacilli is comparatively rarely recorded. Another disturbing element is the known fact that certain organisms act very differently, according to the tissues in which they develop. The pneumococcus in the lung may produce a lobar pneumonia which is quite different from the infectious conditions of the neck in which it may be the only demonstrable organism. The streptococcus of cutaneous erysipelas seems to act in an entirely different manner from the same organism in the deep tissues of the neck.

In twelve cases in which the character of the infection

is noted the following organisms were found: Case 1, streptococcus; 2, staphylococcus and pneumococcus in the mouth and streptococcus in the pus; 3, pure streptococcus; 4, Eberth's bacillus in the spleen and pure streptococcus in the tissues; 5, pneumococcus and some streptococcus; 6, streptococcus; 7, staphylococcus albus and aureus; 8, pneumococcus; 9, streptococcus; 10, large bacillus and staphylococcus aureus and pyogenes; 11, some bacilli, diplococci and streptococci; 12, staphylococci, streptococci and some non-identified organisms.

We thus see that in Cases 1, 2, 3, 4, 6 and 9 streptococcus in the pus and tissues was the sole organism detected. In Case 8, pneumococcus alone was found. In Case 7, staphylococcus alone was found. So that it appears that the same clinical affection can be produced by at least three different organisms. Continuing, we find in some cases the pneumococcus associated with streptococcus (Case 5), with staphylococcus (Case 2), and with streptococcus and some bacilli in Case 11. In Case 12, staphylococcus is found associated with streptococcus and other non-identified organisms. All of which tends to show that these septic neck infections may arise either from a single, but not always the same, variety of organism, or may be a mixed infection of so great complexity as to be impossible of exact identification. Lockwood[3] who gave considerable study to the question of infection states that Ludwig's angina is probably a mixed infection of the most complex kind. That this is so is true in some cases, but in many only a single organism, usually streptococcus or pneumococcus, has been found even in typical cases. From these facts we must conclude that the septic infections of the neck which include those classed as Ludwig's angina can be caused either by one of several organisms, as the staphylococcus, streptococcus and pneumococcus, or by a mixture of various forms, including a gas-producing bacillus.

2. How do the infecting organisms gain access to the tissues?

In many cases the mode of access is unknown, but it is practically established that the infection starts from some lesion in the mouth or throat. Most often it starts from the teeth. In many cases trouble with the teeth antedates or coincides with the onset of the infection. In some cases (as in Case III.) the infection follows so rapidly as to leave no doubt as to the causal relation. As pointed out by Semon the infection does not usually involve the nasal cavities. C. J. Aldrich[4] describes a case that almost certainly started from the tonsil, and this organ has been frequently found involved and another[5] following a pin scratch of the frænum. He also suggests that the infection involves the salivary glands by being transmitted along their ducts. W. O. Humphrey[6] also describes a case preceded by tonsilitis. A case by C. M. Harris[7] suggests an inflammation of the middle ear as being a starting point. The question of mode of origin may be an extremely important one, particularly to dentists. In one of my cases (Case III.) a young lady had an abscess on a lower molar tooth. She went to a dentist who injected a solution of cocaine around the tooth and extracted it. Swelling followed almost immediately and soon assumed the character of Ludwig's angina. It was incised on the fourth day, recovery following. In this case the dentist was accused of having started the inflammation by the use of infected solution or instruments. He stated this was not the fact, as he had used all possible antiseptic precautions. In such a case it is practically impossible for a dentist to demonstrate his innocence. The frequency of infection following trouble with the teeth is such that when a high grade of inflammation exists and an abscess may be forming or already formed dentists will neither extract the offending tooth, nor open the abscess, nor attempt any operative means of relief for fear they should be held accountable for subsequent results.

3. What tissues are attacked?

It is evident that the parts attacked will depend to a certain extent on the location of the breach or injury at

which the infection entered. Semon cites many cases in which the focus of the inflammation involved the tonsil, epiglottis and larynx. In some instances the discharge breaks into the larynx. Œdema of the glottis not infrequently necessitates tracheotomy and may lead to death. When the teeth are the starting point the inflammation involves the periosteum of the lower jaw and thence invades all the surrounding tissues. In many instances (as in Case VI.) the exact point of commencement is unknown and attention is first attracted by the swelling of the tissues of the floor of the mouth and beneath the jaw. While the point at which the infection starts localizes the disease at its commencement, it progressively spreads and involves all the tissues within its scope. No matter how it commences, it spreads along the connective tissues by direct continuity. It is not transmitted by the lymphatics. The lymphatic glands do not become enlarged by infection carried to them by the lymph-stream from the infectious focus, but they are involved in the infected connective tissue surrounding them. In many cases the deep tissues are markedly involved causing a peculiar "wood-like" induration and yet there may be but little or no redness of the skin. This is particularly true early in the course of the disease. As it progresses all the tissues become affected. The bone becomes bare and the soft parts become gangrenous. It is a gangrene of the deep-lying connective tissues and the muscles within them. The process seems to experience difficulty in piercing the deep fascia, hence the skin and subcutaneous tissue are often but little affected. Commonly, particularly early in the disease, there is but little tendency to the formation of pus, and when the epiglottis and larynx are involved, œdema supervenes and causes suffocative symptoms. Early incisions often give exit only to serum and no pus is found. It usually makes its appearance later and is dark colored and peculiarly offensive.

4. How the infection progresses.

As already stated it progresses by direct contiguity of

tissues. Sometimes it begins on one side below and behind the angle of the jaw and passes directly across to the opposite. At other times it passes downward on the neck as far as the clavicles and sternum (Case V.). If it is inside, it soon involves the larynx and tissues around the œsophagus and difficulty in swallowing and breathing may be early symptoms. In fatal cases it follows the cervical tissues down the neck and into the mediastinum and produces a septic pneumonia. The progress of the disease is comparatively acute, often running its course in six to twelve days. It may stop at any time, or it may progressively increase until death is caused by septic infection. In laryngeal cases, death may occur early from suffocation.

5. How it influences the parts locally.

This has already been detailed to a considerable extent. Swelling is the first sign. It shows itself under or behind the lower jaw, in the floor of the mouth, pushing the tongue toward the roof, or in the larynx and epiglottis in the form of œdema. The skin often is normal in color, especially early in the affections and there may be no tendency to the formation of abscesses. In infections from pyogenic organisms, the skin becomes red and rarely considerable collections of pus may occur. It is frequent for openings to occur alongside of the teeth posteriorly and foul, ichorous, pus to exude into the mouth. Later the tissues become gangrenous and may come away as sloughs if the patient survives. If the disease tends to recovery, the local conditions improve with great rapidity, and usually leave no serious results.

6.—How is the system affected?

The affection is primarily a local one, and the general system only becomes involved later. The fever for several days may be moderate, about 101°, but later when sepsis is marked rises to 105° or 106°. These very high temperatures are exceptional. Especially when the streptococcus is the prevailing infecting organism the temperature may not rise above 101° or 102°, even though the case is tending to a fatal issue. In cases of mixed infections the presence of

pyogenic organisms (staphylococci, etc.), may cause the temperature to run higher. At the commencement there is practically no systemic depression but in a few days it becomes marked and deepens to the end. Death may occur either comparatively early from suffocation or heart failure, or later from exhaustion and sepsis. When the disease attacks the larynx death may occur suddenly and before the supervention of marked septic depression. Whether these deaths are due to suffocation or heart failure caused partly by sepsis and partly by the impeded respiration is sometimes difficult to say.

In one case (Lombard et Caboche[8]) a patient who had had great difficulty in breathing was talking with his wife when on reaching for his handkerchief he suddenly fell over and quickly expired. Case No. IX. died in almost the same manner.

In Robertson and Biedert's[9] case sudden death occurred after a tracheotomy had been performed, so that suffocation could not have been the cause. In one of Ross' [10] cases likewise sudden death resulted while the opening existing through the larynx was sufficient to preclude respiratory obstruction.

In Case VIII. the dyspnœa was so great as to require tracheotomy and the patient died on the table. These sudden deaths occur usually in patients in which the epiglottis and larynx are affected and the dyspnœa marked. Involvement of the larynx is indicated by the diminution and loss of voice and difficulty in respiration.

The question of the affection being epidemic has been suggested by Seymour Taylor,[11] who saw a series of cases in the Hammersmith district. F. Murchison[12] and Klein[13] also refer to an outbreak in the Hebrides. Klein states that it is not contagious as there were never two cases in the same family. Five of my own cases came from a single section of the city in a period of five weeks. Thus it is seen that while it can hardly be said to occur in epidemics like the infectious fevers, still it does occur sometimes

in groups, and more frequently than at others, as is the case with that other streptococcus-infectious disease, cutaneous erysipelas.

The question of its infectious character is likewise of importance.

Two of my cases resembled erysipelas so much that they were isolated. In fact it would be best if all these cases were isolated. In most instances it is largely a streptococcus infection and acts like erysipelas. Even the cases that show pneumococcus and staphylococcus infection act clinically much like the others and seem to be but little less virulent. Some have been inclined to regard the disease as being a true erysipelas, but this term could hardly be applied to those cases showing only staphylococci or pneumococci. The disease also acts at times like a common pyogenic affection, all signs ameliorating as soon as an incision is made and tension relieved. Would anyone expect a cutaneous erysipelas to act so? It suggests the possibility of curing the latter disease at once by making free incisions into the affected area.

Another character of the disease is that it sometimes shows a tendency to again extend after apparently convalescing. This occurred in three of my cases and in Dr. Ross' case death occurred suddenly two weeks after the case was reported, and the autopsy showed ulceration of the larynx.

Diagnosis.—The question of diagnosis is intimately associated with that of treatment. It is one not so much of character as it is of degree, but even the question of character may be obscure. Many practitioners have never seen nor recognized a bad case of the so-called Ludwig's angina; as a consequence, in its early stages particularly, it is apt to be unrecognized and energetic treatment deferred until too late. Statistics are practically useless. A septic infection is dangerous according to its extent, and these infections occur in all grades. If mild cases are seen the mortality is slight and if serious cases are seen the

mortality is high. In the ten cases here recorded four died, a mortality of 40 per cent. It must be borne in mind, however, that many light cases which recover are not considered to be of the kind we are now discussing. In making a diagnosis of inflammatory and œdematous affections of the throat and neck, it should be borne in mind that one class of cases as already stated is local in character and usually remain local and do not show the same tendency to spread through the medium of the connective tissues as do the other. Those arising from mechanical and chemical irritants, from interference of the blood-supply producing œdema; from surface inflammations as glossitis, stomatitis, pharyngitis, laryngitis; from inflammations of neighboring organs as the tonsils, salivary and lymphatic glands, syphilitic and tuberculous ulcerations may all be confounded with acute septic infection. In some cases it is impossible to draw the distinguishing line particularly in the early stages, yet it is essential that the true character be recognized as soon as possible, because one class tends to pursue a comparatively benign course while the other pursues a decidedly dangerous one.

The onset of the affection is often insidious, yet some cases are fulminating. That of Biedert and Robertson completed its fatal course in ten hours.

The disease produces local signs before general symptoms, and attention may first be attracted by a swelling which may be either below the jaw in the submaxillary region or posteriorly over the parotid region. The hard "board-like" character of the swelling is almost pathognomonic. Sometimes the skin is pale, sensitiveness not marked, and the temperature raised but one or two degrees. In other cases the skin may be a dusky red, tender, hard and painful to the touch, and the temperature high, 102° or 103°. Swelling of the floor of the mouth pushing the tongue upward to the roof and forward, with difficulty in swallowing and some difficulty in breathing, are early noticed. Chills may occur and dirty offensive pus may

break into the mouth near the molar teeth. The swelling may extend down to the clavicle and up on the temple and a large abscess may form beneath the lower jaw. The temperature rises and death from sepsis follows usually inside of twelve days. Death may occur early from involvement of the larynx, this involvement being indicated first by a hoarseness of the voice and then by its loss. The progressive involvement of the deeper tissues should settle at once the question of diagnosis.

Treatment.—I am firmly convinced that the disease in its early stage is a purely local affection whose extension can be promptly cut short by fearless surgical treatment. Procrastination and timidity as well as a failure to recognize the dangers of delay, are the undoubted causes of the loss of many cases. Fears of unnecessarily scarring the patient or of encountering alarming hæmorrhage both suggest delay. He who waits for the formation of pus before incising waits too long. When a case presents itself with a hard board-like swelling beneath the jaw it is evidence of probable cellular-tissue infection. Administer primary anæsthesia with ethyl chloride, ether or chloroform, and make an incision in the median line between the symphysis and the hyoid bone and carry it through all the tissues; better all the way into the mouth, at least until the point of the knife can be recognized by the finger inside the mouth beneath the tongue. This incision is easily made, devoid of danger, is accompanied by no hæmorrhage, and drains effectively the infected area. If it is made early no pus will be found but only blood, or a little thin serum. The relief, however, is immediate. If the swelling is more toward the angle of the jaw, or in the parotid region, then incise the skin and with a pair of hæmostatic forceps bore slowly into the swollen tissues, expanding the blades and if necessary inserting drainage tubes. In very bad cases the larger the incisions the better, and one or two of my own could probably have been saved had this been done instead of relying on drainage tubes. In this affection pus does not often show a ten-

dency to accumulate, and the large incision relieves tension and allows the gangrenous tissues to be cast off.

In œdema of the epiglottis and larynx, ice and inhalations (spray) of cocaine and adrenalin may be of service, but tracheotomy should not be deferred too long. A high tracheotomy is probably just as efficient as a low one, and much less dangerous. In one of the cases here recorded death ensued on the table from hæmorrhage, and this is hardly to be wondered at when we recall the vessels which may be encountered. The large distended inferior thyroid veins, an anomalous thyroid artery, an innominate slightly more to the left than usual, or a high left innominate vein crossing above the top of the sternum, may any one of them cause a fatal issue.

CASES.

CASE I.—Young woman, aged 22. Was admitted for a swelling of face and jaw. An examination of the mouth failed to reveal any cause for the infection. There was no evidence of carious teeth, tonsilitis or other focus of infection. About a week previous to admission she noticed that the side of her face was swollen, principally behind the angle of the jaw. It rapidly involved the whole neck and both sides of the face. It was slightly red, hard and somewhat painful. It had been poulticed. She could hardly swallow, the voice was altered and hoarse, the tongue swollen, and the jaws could be separated only a half inch. Temperature was 101.3°.

An incision was made in the median line beneath the chin, extending into the mouth, and another behind the angle of the jaw. No distinct pus was found but the next day very thick, offensive pus discharged. The swelling and temperature rapidly diminished and the discharge had almost ceased by the twelfth day, when her temperature rose to $100\frac{3}{5}°$ and the swelling again returned to again disappear after a few days.

An examination of the pus showed it to be a pure streptococcus infection. (See Fig. I.)

CASE II.—A Russian, male, aged 26, was brought to the Episcopal Hospital with the history of having had several teeth extracted from the back part of the left lower jaw. About

FIG. 1.

four hours later the jaw became swollen and two days afterwards the right side became swollen and painful. On admission, he was unable to speak English, both jaws were swollen, breath was fœtid, and stinking pus was escaping into the mouth from the left molar region. The patient looked very ill. Tongue was coated. Urine 1026, with marked reaction of albumen. An incision below the angle of the jaw gave exit to a small quantity of foul pus. He swallowed with difficulty, his respiration became jerky and hurried, varied from 102° to 104° and once went to 106°. He died of sepsis nine days after admission.

The color of the skin in this case was pale rather than red, and at no time was there marked evidences of any accumulation of pus. It is barely possible that more free incisions would have benefited this case.

CASE III.—A young woman, aged 24 years, had a lower right molar tooth extracted for an abscess of its roots. The dentist injected cocaine into the gums. Within an hour after the extraction the cheek began to swell. On the next day cold was applied and a mouth-wash used. The day following she was somewhat better, but on the fourth day the swelling got worse and the pain increased. Leeches and ice were applied but on the day following the swelling extended from ear to ear around under the jaw; it was tender, a little red and quite brawny, and hard to the touch.

An incision was made between the hyoid bone and the symphysis, extending to the mucous membrane of the mouth just below the tongue. No pus was obtained.

During the night the patient had considerable difficulty in breathing, but in the morning a free discharge of pus made its appearance and immediate relief followed.

In six days after incision the discharge of pus ceased and she was practically well. Her temperature during the attack ranged from 101° to 102.8°.

The dentist stated that he used a fresh solution of cocaine and sterilized instruments in injecting it.

The immediate following of the inflammation after

the extraction looks like cause and effect and the course after incision demonstrates its efficacy.

CASE. IV.—A young man, aged 19; had, two weeks prior to admission, pain in the teeth and swelling of the jaw. The last molar on the affected side was decayed. The swelling began at angle of the jaw. He could hardly open his mouth. Urine Sp. Gr. 1027; trace of albumen; no sugar; no casts. Temperature 104.6°. An incision was made below the angle of the left side of the jaw and considerable pus was evacuated. About two weeks later the left side again became swollen, his temperature rose and he looked ill. The swelling was red and indurated and the breath foul. A small amount of pus escaped from the original incision. The symptoms gradually abated and in six days he was discharged cured.

It will be observed that this patient also had a relightening up of the trouble after the subsidence of the first attack.

CASE V.—A young man, aged 20; had for some time a bad tooth in the right side of the lower jaw. Eighteen days prior to admission the right side of the neck began to swell and on admission the neck was enormously swollen extending from the zygoma above to the clavicle below, and from the right ear around the neck to beyond the median line. Voice hoarse; could not breathe lying down, and had marked difficulty in swallowing. His temperature was 101°, pulse, 120, respirations 24. General condition, good.

Two incisions were made, one in the median line and the other beneath the angle of the jaw. Practically no pus was obtained. He had to sit up all night, but his dyspnœa gradually disappeared. Two days later there was a small amount of pus, which showed streptococcic infection.

On the fifth day his temperature was normal and on the tenth day he was discharged with the wounds not yet closed.

Another example of the efficacy of free incisions and deep exploration with a hæmostatic forceps. (See Fig. II).

CASE VI.—A man, aged 23 years, was admitted to the hospital for typhoid fever, having been ill two weeks. Seven days after entrance while feeling much better it was noticed in the

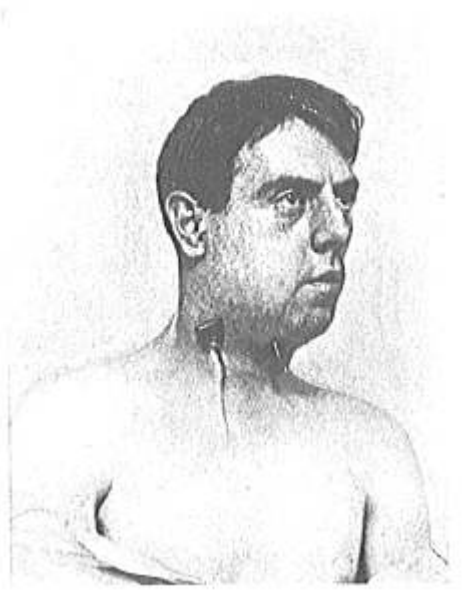

FIG. 2.

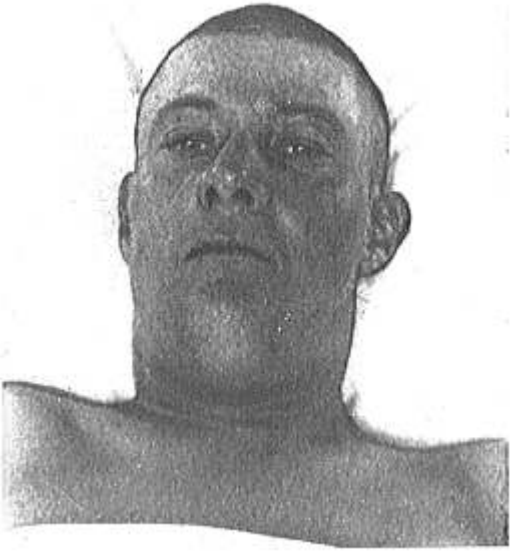

FIG. 3.

morning that his face looked fat. By three o'clock in the afternoon the neck was swollen, hard and tender in the submental region. He could only open his mouth half way. Leucocytosis of 12000. No growth on the tonsils. The next day swelling was more marked and indurated nearly to the sternum. He was beginning to have difficulty in respiration. An incision was made through the swollen parts between the symphysis and hyoid bone, extending through into the mouth. A considerable amount of thin brownish-green discharge escaped. Cultures of streptococci, staphylococci and pneumococci were obtained from the mouth and streptococci and staphylococci from the wound discharge. The day following the wound was discharging pus freely and pus was also discharging from the right ear. The next day he was still better but the following day the neck again swelled, became red and looked like erysipelas. He then passed through a regular attack of cutaneous erysipelas, which lasted three weeks. It spread up over the left side of the face and closed the left eye. Then the wound healed but the right side of the face began swelling, involving the left eyelids. An abscess formed in the left eyelid and discharged gray pus. An abscess formed in the left molar region and was opened, and the left ear also discharged pus. The erysipelas finally disappeared and the patient recovered. (See Fig. III.)

This was a case of mixed infection. The erysipelas was a typical attack and I regard it as not being a new infection from without but a direct extension by continuity of tissue of the streptococcus infection which began in the submental region. A case like this goes far to prove that the original infection was much the same as occurs in ordinary erysipelas. This is the third case in the series in which a subsidence of the original attack was followed by a secondary outbreak.

CASE VII.—A man aged 48 years had been attending the outpatient department of St. Joseph's Hospital for an infected wound of the finger. He was absent for some time and was returned to the hospital in such a septic and depressed state that no history could be obtained. The left side of his face in the parotid region and behind the angle of the jaw was hard

and swollen but not very red; œdematous and tender on pressure.

The mouth was partly open, tongue coated, teeth in bad condition and a foul discharge of pus along the posterior portion of the left lower molar teeth. The index-finger of the right hand contained a small quantity of pus. Heart and lungs negative. Urine 1020, acid; albumen 4 per cent. ; no sugar.

Temperature on admission 98.6°; next day it went to 106.4°. It varied between these extremes with chills until he died on the sixth day after admission. The swelling invaded the temporal region and rales appeared in the lungs and then he died from sepsis. He was treated by incisions and a large drainage-tube from the angle of the jaw into the mouth.

This case was pyæmic on admission, and it is doubtful if any treatment would have saved him; but it is probably worth while in such cases as this to make a long incision from below the ear, behind the angle of the jaw and as far forward as the swelling extends. I believe drainage by means of tubes is insufficient and wide-open incisions are required.

Case VIII.—This case I saw but did not treat. He was a man, aged 35, an engineer, who was brought into the hospital drunk. He had a swelling beneath the jaw and was transferred to the surgical wards. The swelling rapidly increased, accompanied by attacks of dyspnœa.

The mouth and throat was sprayed with a solution of cocaine, adrenalin and menthol. On the fourth day he had such a severe attack that tracheotomy was attempted but he died on the table from hæmorrhage.

When death occurs from suffocation it is usually not as in this case from an acute paroxysm but more usually by a gradual shutting off of the air until the overloaded heart simply gives out. This case illustrates the difficulties and dangers of performing tracheotomy when the neck is greatly swollen.

CASE IX.—Was admitted under the care of my colleague, Dr. Edsall. It was a man, aged 42 years. He retired one night apparently well, but was awakened the next morning being scarcely able to breathe and having a violent rigor. He could only lie a short time and then had to sit up. He felt as if there was a lump in the throat. He stated that there had been a lump on the right side of the throat, which broke and allowed a lot of foul material to run down his throat. On admission he was short of breath and had a hard mass on the left side of the neck. No fluctuation or other evidences of suppuration. Uvula much swollen and œdematous. Tonsils could not be seen. The swelling was incised but no pus was obtained. Thorough drainage by means of a tube was employed, which gave some relief. Urine 1023, acid marked, trace of albumen, no sugar. He died apparently of suffocation, suddenly, on the second day after admission and the fourth day of the disease.

Another instance of the apparent inefficiency of the drainage-tube.

CASE X.—Was under the care of my colleague, Dr. Frazier. It was that of a man, aged 61 years. A decayed loose tooth had been occasioning the patient some trouble and a week before admission he had had considerable pain in the lower jaw. A swelling began under the jaw, which was painful and very hard. He had difficulty both in breathing and swallowing.

On admission there was a hard swelling under the chin, extending down to the larynx. The breath was extremely offensive. There was redness and swelling under the tongue. The loose tooth from which the trouble originated was still in. The swelling was incised and a thin watery material oozed out, along with blood. There was no free pus. Three openings were made and two rubber tubes were put in as drains and a hæmostat was thrust in several directions and opened and drawn out. Very little thin, watery, offensive fluid escaped. Considerable of this same foul-smelling material was later seen on the dressings. Considerable relief was obtained from the operation. At one time the patient was expected to die, but eventually recovered after a stay in the hospital of nine days.

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